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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/234,208	01/20/1999	JONI KRISTIN DOHERTY	49321-1	6406
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DAVIS WRIGHT TREMAINE, LLP 2600 CENTURY SQUARE 1501 FOURTH AVENUE SEATTLE, WA 98101-1688			EXAMINER UNGAR, SUSAN NMN	
			ART UNIT 1642	PAPER NUMBER

DATE MAILED: 11/03/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

09/234,208

Applicant(s)

DOHERTY ET AL.

Examiner

Susan Ungar

Art Unit

1642

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☐ Responsive to communication(s) filed on 24 August 2005.
- 2a) ☐ This action is FINAL. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☐ Claim(s) 1-3, 8-10, 18-20, 27-30 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) 27 is/are allowed.
- 6) ☐ Claim(s) 1-3, 8-10, 18-20 and 28-30 is/are rejected.
- 7) ☐ Claim(s) 20 is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- * See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date 8/24/05
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: _____

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to you 37 CFR 1.114. Applicant's submission filed August 24, 2005 is acknowledged and has been entered.
2. The Amendment filed August 24, 2005 in response to the Office Action of February 24, 2005 is acknowledged and has been entered. Previously pending claims 3, 10, 18 have been amended. Claims 1-3, 8-10, 18-20, 27-30 are currently being examined.
3. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

New Grounds of Objection

4. Claim 20 is objected to because it does not further limit claim 19 from which it depends, but rather broadens the claimed invention. This objection can be obviated, for example, by amending the pendency of the claim so that it depends from claim 18.
5. The amendment filed August 24, 2005 is objected to under 35 U.S.C. 132 because it introduces new matter into the specification. 35 U.S.C. 132 states that no amendment shall introduce new matter into the disclosure of the invention. The added material which is not supported by the original disclosure is as follows:

Applicant's submission of a Sequence Listing and CRF is objected to because a review of position 31 in the originally filed sequence listing discloses that residue 31 is a Lys and not the Met as currently amended. A review of position 86 of the originally filed sequence listing reveals that residue 86 is an Arg,

not the Ile as currently amended. A review of position 148 of the originally filed sequence listing reveals that residue 148 is a Leu not the Ile as currently amended. A review of position 198 of the originally filed sequence listing reveals that residue 198 is a Cys not a Met as currently amended. A review of position 282 reveals that residue 182 of the originally filed sequence listing is a Pro not a Met as currently amended. Finally a review of residue 303 of the originally filed sequence listing reveals that residue 303 is a Leu, not the Tyr as currently amended.

Applicant is required to cancel the new matter in the response to this Office action.

New Grounds of Rejection

Claim Rejections –35 USC 112

6. Claims 1-2, 8-9, 18-20, 29-30 are rejected under 35 USC 112, first paragraph because the specification, while being enabling for an isolated peptide consisting of SEQ ID NO:1 or comprising SEQ ID NO:2 does not reasonably provide enablement for any isolated peptide comprising SEQ ID NO:1, having from about 50-79 or 69-79 amino acids taken from SEQ ID NO:1 or from about 80-419 or about 350-419 amino acids from SEQ ID NO:2 which bind to the extracellular domain of HER-2. The specification does not enable any person skilled in the art to which it pertains or with which it is most nearly connected to make the invention commensurate in scope with these claims.

The claims are drawn to any isolated peptide which comprises SEQ ID NO:1, has about 50-79 or 69-79 amino acids taken from SEQ ID NO:1 or about 80-419, or about 350-419 amino acids taken from SEQ ID NO:2 which binds to the extracellular domain of HER-2. Thus, to meet the limitations of the claims, the peptides must bind to a specific region of the HER-2 peptide. The specification

discloses a single isolated polypeptide, p68HER-2, which comprises a truncated extracellular domain of HER-2 which is extended by 79 amino acids, domain ECDIIIa. Both p68HER-2 and ECDIIIa bind to p185HER-2 and do not activate signal transduction. The specification provides no objective evidence that any other isolated polypeptides would function as ECDIIIa and p68 HER-2 do. This means the claims are drawn to truncations of ECDIIIa at either the 3' or the 5' end, or both and truncations of p68HER-2 at the 3' end, wherein, the effects of the truncations cannot be determined from the information in the specification

One cannot extrapolate the teaching of the specification to the scope of the claims because the claims are drawn to truncations of ECDIIIa at either the 3' or the 5' end, or both and truncations of p68HER-2 at the 3' end wherein the specification does not teach the amino acids critical for binding to HER-2 or the effects of binding to HER-2 of the truncation of p68HER-2 and the specification has not enabled all of these types of modified polypeptides.

In particular, protein chemistry is probably one of the most unpredictable areas of biotechnology. For example, Bowie et al (Science, 1990, 257:1306-1310, IDS item) teach that an amino acid sequence encodes a message that determines the shape and function of a protein and that it is the ability of these proteins to fold into unique three-dimensional structures that allows them to function and carry out the instructions of the genome (col 1, p. 1306). Bowie et al further teach that while it is known that many amino acid alterations are possible in any given protein, the position within the protein's sequence where such amino acid alterations can be made with a reasonable expectation of maintaining function are limited. Certain positions in the sequence are critical to the three dimensional structure/function relationship and these regions can tolerate only conservative alterations or no

alterations. Residues that are directly involved in protein functions such as binding of p68HER2/ ECDIIIa will certainly be among the most conserved (see Bowie et al, Supra, (col 2, p. 1306). However, the specification provides no guidance on structure or residues that are critical to the binding function of the invention as claimed. The artisan is left to random experimentation in order to determine which residues may be deleted in order to produce an altered polypeptide that will function as claimed and contemplated. Random experimentation is undue.

Although it appears that binding specificity of the claimed invention resides in the 79 amino acid extension, ECDIIIa, the exquisite sensitivity of binding proteins to alterations of even a single amino acid is well known in the art. For example, although drawn to the antibody arts, the following is clearly relevant to the 185HER2 binding function of the claimed invention. Rudikoff et al, (PNAS, USA, 1982, 79: 1979) specifically teach that even minor changes in the amino acid sequence of the heavy and light variable regions, particularly in the CDRs, may dramatically affect antigen-binding function. In particular, Rudikoff et al teach that alteration of a single amino acid in the CDR of a phosphocholine-binding myeloma protein results in the loss of antigen-binding function. Further, the sensitivity of binding proteins to alterations of even a single amino acid in a sequence are exemplified by Burgess et al (J of Cell Bio. 111:2129-2138, 1990, IDS item) who teach that replacement of a single lysine residue at position 118 of acidic fibroblast growth factor by glutamic acid led to the substantial loss of heparin binding, receptor binding and biological activity of the protein. These references demonstrate that even a single amino acid alteration or what appears to be an inconsequential chemical modification will often dramatically affect the biological activity and characteristics of a binding protein. Given the above, as

drawn to SEQ ID NO:1, given that the residues of ECDIIIa that are critical to binding have not been identified, it could not be predicted which truncated version of SEQ ID NO:1 would function as claimed and one would not know how to make the claimed invention. Although Applicant might argue that one of ordinary skill could screen for the species that would function as claimed, in particular, screening assays do not enable the claimed invention because the court found in (*Rochester v. Searle*, 358 F.3d 916, Fed Cir., 2004) that screening assays are not sufficient to enable an invention because they are merely a wish or plan for obtaining the claimed chemical invention. Further as drawn to truncation of both SEQ ID NO:1 and SEQ ID NO:2, the effects of truncation on these polypeptides also cannot be predicted because the truncations would be expected to alter the configuration of the polypeptides and this alteration of configuration would be expected to affect the conformation of the binding site, even if the binding site were not to be deleted. Given the exquisite sensitivity of binding proteins, it would not be expected and could not be predicted that the invention would function as claimed in the absence of further guidance. Finally, as drawn to polypeptides comprising SEQ ID NO:1, the claims are drawn to polypeptides that include unlimited amino acid residues on both the C and N termini of the polypeptide. Given the teaching of Bowie et al that the amino acid sequence encodes a message that determines the shape and function of a protein and that it is the ability of these proteins to fold into unique three-dimensional structures that allows them to function and carry out the instructions of the genome, it is clear that the folding and shape of the undefined polypeptide cannot be predicted. Since the claim requires that the polypeptide bind to the extracellular domain of HER-2 neu, it is clear that the claims require that the sequence be found on the outer surface of

the claimed polypeptide, but given the undefined nature of the polypeptide, even with SEQ ID NO:1 present in the undefined polypeptide, alterations in protein folding due to protein sequence additions could mask SEQ ID NO:1, and it could not be predicted whether SEQ ID NO:1 would be found on the surface of the polypeptide. The specification provides neither information nor guidance on how to make the broadly claimed polypeptide. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention would function as broadly claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

7. If Applicant were able to overcome the rejection set forth above, Claims 18-20, 29-30 would still be rejected under 35 USC 112, first paragraph because the specification, while being enabling for a pharmaceutical composition for treating solid tumors that overexpress HER-2 comprising SEQ ID NO:2, does not reasonably provide enablement for a pharmaceutical composition for treating solid tumors that overexpress HER-2 consisting of SEQ ID NO:1, comprising SEQ ID NO:1, comprising the claimed fragments of SEQ ID NO:1 or SEQ ID NO:2. The specification does not enable any person skilled in the art to which it pertains or with which it is most nearly connected to practice the invention commensurate in scope with these claims.

The claims are drawn to pharmaceutical compositions for the treating of solid tumors that overexpress HER-2. The specification teaches that both SEQ ID NO:1 and SEQ ID NO:2 bind p185HER-2 and that neither of the polypeptides

stimulates phosphorylation of p185HER-2 (p. 6). The specification exemplifies the isolation of SEQ ID NO:2 from SKBR-3 cells which were resolved by SDS-PAGE in 7.5% acrylamide gels and analyzed as a Western blot (p. 6). Using specific antibodies either for the novel 79 unique amino acid residue or the C-terminal sequence, SEQ ID NO:1, a 68 kDa product was identified. This 68 kDa protein product is an alternative HER-2 transcript in cell extracts and in extracellular media from several cell lines (p. 7). The specification further teaches that since SEQ ID NO:2 occupies but does not activate, it could block dimerization of p185Her-2. Further, since SEQ ID NO:2 contains subdomains I and II of p185HER-2, the specification hypothesizes that since subdomain I may be the low affinity, promiscuous ligand binding site required for recruitment of p185Her-2 into heteromeric complexes, SEQ ID NO:2 could block this site and therefore obstruct recruitment of p185HER-2 into heteromeric complexes and consequently the recruitment of p185HER2 into dimmers. The specification also suggests that p68Her-2 could compete with an uncharacterized ligand for binding to p185HER-2 (p. 9). The specification teaches Western blot analysis to determine whether SKBR-3 cells, which express the alternative sequence in its cDNA produced a protein that reacts with anti-ECDIIIa antibody. A 68 kDa protein protein was identified that bound to both anti-ECDIIIa antibody and antibody to extracellular domain of p185HER2 (pages 17 and 18).

One cannot extrapolate the teaching of the specification to the scope of the claims because the composition is specifically claimed for the treatment of solid tumor and Gura (Science, 1997, 278:1041-1042) teaches that researchers face the problem of sifting through potential anticancer agents to find ones promising enough to make human clinical trials worthwhile and teach that since formal

screening began in 1955, many thousands of drugs have shown activity in either cell or animal models but that only 39 have actually been shown to be useful for chemotherapy (p. 1041, see first and second para). Because of the known unpredictability of the art, in the absence of experimental evidence, no one skilled in the art would accept the assertion that the truncated SEQ ID NO:2 or SEQ ID NO:1 or its fragments which specifically binds to p185 could be predictably used as an anti-cancer agent for cancer therapeutic strategies as claimed. Further, the refractory nature of cancer to drugs is well known in the art. Jain (Sci. Am., 1994, 271:58-65) teaches that tumors resist penetration by drugs (p.58, col 1) and that scientists need to put expanded effort into uncovering the reasons why therapeutic agents that show encouraging promise in the laboratory often turn out to be ineffective in the treatment of common solid tumors (p. 65, col 3). Curti (Crit. Rev. in Oncology/Hematology, 1993, 14:29-39) teaches that solid tumors resist destruction by chemotherapy agents and that although strategies to overcome defense mechanisms of neoplastic cells have been developed and tested in a number of patients, success has been limited and further teaches that it is certainly possible that cancer cells possess many as yet undefined additional molecular mechanisms to defeat chemotherapy treatment strategies and if this is true, designing effective chemotherapeutic regimens for solid tumors may prove a daunting task (para bridging pages 29-30) and concludes that knowledge about the physical barriers to drug delivery in tumors is a work in progress (p. 36, col 2). It is clear that based on the state of the art, in the absence of experimental evidence, no one skilled in the art would accept the assertion that the truncated SEQ ID NO:2 or SEQ ID NO:1 or its fragments which specifically binds to p185 could be predictably used as an anti-cancer agent for cancer. It is noted that although

Applicant previously submitted Declarations which exemplified the *in vivo* efficacy of a polypeptide consisting of SEQ ID NO:2, this information is not commensurate in scope with the claimed invention. It is clear from the information in the specification that the mechanism of action of SEQ ID NO:2 is unknown and it can't be predicted from the information in the specification or the art of record that polypeptides other than SEQ ID NO:2 would function as claimed. The specification provides neither information nor guidance on how to use the broadly claimed polypeptide. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention would function as broadly claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

8. Claims 18, 20 and 30 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to

The claims are drawn to a pharmaceutical composition for treating solid tumors comprising a monoclonal antibody that binds to the extracellular domain of p185HER2 combined with SEQ ID NO:2 or a fragment of SEQ ID NO:2.

The specification teaches that SEQ ID NO:2 comprises the extracellular domain of p185HER2 and a 79 amino acid insert.

One cannot extrapolate the teaching of the specification to the enablement of the claims because it would be expected that the monoclonal

antibody to the extracellular domain of p185HER2 would bind to SEQ ID NO:2 which comprises the extracellular domain of SEQ ID NO:2 or the portion of SEQ ID NO:2 that is not truncated. Given that the antibody would be expected to sequester SEQ ID NO:2 in solution, it is not clear how the complexed antibody/SEQ ID NO:2 would be expected to bind to p185HER2 *in vivo*. In particular, the specification provides neither guidance on nor exemplification of the claimed invention and for the reasons set forth above drawn to Gura, Jain and Curti, one would not know how to use the claimed invention. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the invention would function as broadly claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

9. If Applicant were able to overcome the rejections set forth above, Claims 18, 20 and 30 would still be rejected under 35 USC 112, first paragraph because the specification, while enabling for the claimed pharmaceutical composition for treating solid tumors comprising Herceptin does not reasonably provide enablement for a pharmaceutical composition for treating solid tumors comprising a monoclonal antibody that binds to the extracellular domain of p185HER2. The specification does not enable any person skilled in the art to which it pertains or with which it is most nearly connected to practice the invention commensurate in scope with these claims.

The claims are drawn to a pharmaceutical composition for treating solid tumors comprising a monoclonal antibody that binds to the extracellular domain of p185HER2. This means any antibody to the extracellular domain of p185 HER2.

The specification teaches that Herceptin is a marketed humanized monoclonal antibody that is used for the treatment of cancer and that binds to the ECD of HER-2 (p. 12).

One cannot extrapolate the teaching of the specification to the scope of the claims because implicit in the use of the pharmaceutical composition is the effective use of the composition in the treatment of solid tumors. However, other than the mention of Herceptin in the specification, there is no disclosure of any other monoclonal antibodies effective to treat cancers overexpressing HER-2 and the specification does not teach how to make a pharmaceutical antibody with the properties required for treatment of an individual who had cancer overexpressing p185 so that it will function as claimed. For example, Stancovski, et al (PNAS, USA, 88:8691-8695, 1991) characterized the effects of various antibodies that bind the extracellular domain of ErbB2 upon the growth of tumor cells. Stancovski, et al teach, while some anti-ErbB2 antibodies inhibit tumor growth, at least one of the anti-ErbB2 antibodies actually accelerates tumor growth (page 8693, column 1). This phenomenon was also reported in Lewis, et al (Cancer Immunology Immunotherapy 37: 255-263, 1993). US Patent No. 5,677,171 teaches that not every anti-ErbB2 antibody can be used as effectively as monoclonal antibody 4D5 (col 18, lines 15-23). More specifically, '171 teaches that some anti-ErbB-2 antibodies inhibited growth to a lesser extent than Mab 4D5 while others failed to inhibit growth. Further, Strobel, et al (Gynecologic Oncology 73: 362-367, 1999) teach discordant effects of contacting cancer cells with two

different neutralizing monoclonal antibodies, i.e., antibodies that block the function of the receptor protein to which they specifically bind (abstract). Despite the fact that both anti-receptor antibodies had been shown to block ligand binding to the receptor, Strobel, et al found that only one of the antibodies could be used effectively to block cancer cell adhesion to inhibit malignancy. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict that the broadly claimed method will function as claimed with a reasonable expectation of success. For the above reasons, it appears that undue experimentation would be required to practice the claimed invention.

Claim Rejections –35 USC 102

10. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

11. Claims 1, 3, 8, 10, 19, 28, 29 are rejected under 35 U.S.C. § 102(b) as being anticipated by Scott et al (Mol. Cell, Biol., 1993, 13:2247-2257, IDS item).

The preamble recitation of pharmaceutical composition is merely suggestive of an intended use and is not given weight for purposes of comparing the claims with the prior art. The claims read on the active ingredients *per se*, which are a polypeptide and a pharmaceutically acceptable carrier.

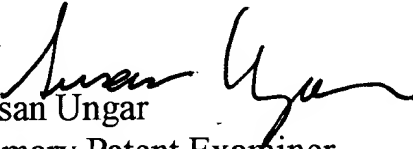
The claims are drawn to an isolated polypeptide comprising SEQ ID NO:1, comprises SEQ ID NO:2.

Scott teaches a concentrate of conditioned medium from SKBR-3 cells, which is a composition comprising a pharmaceutically acceptable carrier (see p. 2249, col 1) which comprises a 68 kDa polypeptide which reacts with polyclonal antibody to the extracellular domain of p185 Her2 (see Figure 5). Although the reference suggests that the 68kDa polypeptide is a nonspecific peptide and therefore is not a truncated HER2 protein because there is almost equal intensity in MCF-7 cells, given that SEQ ID NO:2 is a 68kDa polypeptide, given that it is expressed in SKBR-3 cells and migrates at 68kDa in Western blot, the claimed polypeptide appears to be the same as the prior art polypeptide, absent a showing of unobvious differences. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the product of the prior art does not possess the same material, structural and functional characteristics of the claimed polypeptide. In the absence of evidence to the contrary, the burden is on the applicant to prove that the claimed product is different from those taught by the prior art and to establish patentable differences. See *In re Best* 562F.2d 1252, 195 USPQ 430 (CCPA 1977) and *Ex parte Gray* 10 USPQ 2d 1922 (PTO Bd. Pat. App. & Int. 1989). Further, given that the isolation method of the prior art comprises the same method steps as in the instant invention, in the same population of cells, the claimed polypeptide is anticipated by the isolated polypeptide of Scott et al because the method inherently led to the isolation of the claimed polypeptide. See Ex parte Novitski 26 USPQ 1389 (BPAI 1993).

12. Claim 27 appears to be free of the art and allowable.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Susan Ungar, PhD whose telephone number is (571) 272-0837. The examiner can normally be reached on Monday through Friday from 7:30am to 4pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Siew, can be reached at 571-272-0787. The fax phone number for this Art Unit is (571) 273-8300.


Susan Ungar
Primary Patent Examiner
March 21, 2005